Complete Summary

GUIDELINE TITLE

American Association of Clinical Endocrinologists medical guidelines for clinical practice for the evaluation and treatment of hypogonadism in adult male patients--2002 update.

BIBLIOGRAPHIC SOURCE(S)

AACE Hypogonadism Task Force. American Association of Clinical Endocrinologists medical guidelines for clinical practice for the evaluation and treatment of hypogonadism in adult male patients--2002 update. Endocr Pract 2002 Nov-Dec;8(6):439-56. [77 references]

COMPLETE SUMMARY CONTENT

SCOPE

DISEASE/CONDITION(S)

Hypogonadism

GUIDELINE CATEGORY

Diagnosis
Evaluation
Treatment

CLINICAL SPECIALTY

Endocrinology
Family Practice
Internal Medicine

INTENDED USERS

Physicians

GUIDELINE OBJECTIVE(S)

To assist health-care professionals in medical decision making for determining the most effective methods of diagnosing and treating hypogonadism in adult male patients

TARGET POPULATION

1. Males with primary testicular failure requiring testosterone replacement (hypergonadotropic hypogonadism)
2. Male patients with gonadotropin deficiency or dysfunction who may have received testosterone replacement therapy or treatment for infertility (hypogonadotropic hypogonadism)
3. Aging men with symptoms relating to testosterone deficiency who could benefit from replacement therapy

INTERVENTIONS AND PRACTICES CONSIDERED

Initial evaluation/assessment

1. Comprehensive history
2. Physical examination
    - Pubertal development
    - Amount and distribution of body hair
    - Presence and degree of gynecomastia
    - Testicular exam and measurement
    - Scrotal exam (masses, varicoceles)
    - Prostate examination
3. Laboratory and ancillary studies
    - Testosterone level determination
        - Free testosterone
        - Sex hormone-binding globulin (SHBG) level
    - Gonadotropin level determination
        - Luteinizing hormone (LH)
        - Follicle-stimulating hormone (FSH)
    - Dynamic tests
        - Gonadotropin-releasing hormone (GnRH) stimulation test
        - Clomiphene stimulation test
        - Human chorionic gonadotropin (hCG) stimulation test
    - Prolactin level determination
    - Semen analysis
        - Sperm count, motility, and morphology
        - If azoospermic, perform fructose test
    - Bone densitometry
    - Pituitary imaging
    - Genetic studies/chromosomal analysis
        - Karyotype testing
        - Fluorescent in situ hybridization studies

- Testicular biopsy and scrotal exploration
- Testicular ultrasonography

4. Differential diagnosis

Treatment

1. Testosterone therapy
    - Long-acting intramuscular preparations
    - Short-acting intramuscular preparations
    - Scrotal patches
    - Transdermal patches
    - Transdermal gel
    - Orally administered agents (considered but not generally recommended in the United States)
2. Gonadotropin therapy
    - Human chorionic gonadotropin (hCG) therapy
    - Human menopausal gonadotropin therapy
    - Gonadotropin-releasing hormone (GnRH) pump therapy
    - Follicle-stimulating hormone (FSH) or luteinizing hormone (LH) therapy
3. Gonadotropin-releasing hormone (GnRH) therapy
4. Other treatment considerations
    - Antiestrogen therapy in oligospermia (considered but not recommended)
    - Assisted reproductive technology, including
        - Intracytoplasmic sperm injection
        - Intrauterine insemination
    - Assessment for pituitary tumors and their treatment
    - Treatment of gynecomastia through breast reduction surgery or aromatase inhibitors (e.g., testolactone)
    - Psychological counseling

MAJOR OUTCOMES CONSIDERED

- Accuracy of diagnostic tests for hypogonadism
- Male fertility
- Virilization
- Sexual function, libido, well-being, and behavior
- Bone mass and incidence of bone fractures
- Cardiovascular mortality

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

Not stated

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Not stated

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Not applicable

METHODS USED TO ANALYZE THE EVIDENCE

Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Not stated

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

Fifteen physicians are acknowledged in the guideline document as reviewers.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

Summary

The major objectives of the initial assessment are to distinguish primary gonadal failure (hypergonadotropic hypogonadism with low testosterone and increased follicle-stimulating hormone [FSH] and luteinizing hormone [LH] levels from hypothalamic-pituitary disorders [hypogonadotropic hypogonadism with low testosterone and low to normal FSH and LH levels]) and to make a specific diagnosis. The initial clinical manifestations may vary, depending on whether the onset of the disorder was prepubertal or postpubertal. Men with hypogonadotropic disorders may achieve fertility with gonadal stimulation. Men with hypergonadotropic disorders are treated with testosterone to achieve virilization and are usually, but not invariably, incapable of achieving fertility.

History and Physical Examination

- A history of major medical problems, medications, toxic exposures, fertility problems, and developmental milestones should especially be noted. Low libido, impotence, fatigue, impaired concentration, and sexual dysfunction are important presenting problems and need to be asked about specifically because most men will not seek medical attention for these symptoms alone.
- The degree of pubertal development, eunuchoid proportions, anosmia, hyposmia, gynecomastia, abnormal hair growth and distribution, abnormal genitalia, presence of varicocele, findings on prostate examination, and testicular size and consistency, in particular, are important physical manifestations for differential diagnosis.

Laboratory and Ancillary Evaluation

- Laboratory testing is directed toward determining whether the patient has abnormalities of reproductive hormones and whether the abnormalities are indicative of testicular or hypothalamic-pituitary disease. The initial laboratory testing should include a morning blood sample for testosterone, prolactin, FSH and LH levels. A semen analysis is needed if fertility potential is at issue.
- If testosterone levels are low-normal and the symptoms and signs indicate hypogonadism, the testosterone study should be repeated, and sex hormone-binding globulin (SHBG) or a free testosterone level by equilibrium dialysis should be determined to help diagnose a hypogonadal state because total testosterone levels may be normal in the setting of hypogonadism if the SHBG levels are increased.
- For the diagnosis of hypergonadotropic hypogonadism, FSH is especially important because FSH has a longer half-life, is more sensitive, and demonstrates less variability than LH. Pooled LH samples (three preferred) may help reduce problems with LH variability associated with a short half-life and pulsatile secretion.
- Dynamic testing of the hypothalamic-pituitary-testicular axis should be done by an endocrinologist and reserved for patients in whom the results of baseline diagnostic testing are equivocal, although interpretation of the results of dynamic testing may be poorly defined.
- In acquired hypogonadotropic hypogonadism, a prolactin level and pituitary imaging study should be done to assess the patient for a possible hypothalamic-pituitary disorder such as a pituitary tumor. Testing of the thyroid, adrenal, and growth hormone axes is also indicated.

- Chromosomal analysis should be considered in men with prepubertal-onset hypergonadotropic hypogonadism to evaluate for Klinefelter's syndrome and related disorders.
- Bone densitometry should be done in men with chronic, untreated hypogonadal disorders to aid in decision making about treatment options to prevent and treat osteoporosis.
- Testicular ultrasonography should be done in patients with clinical findings suggestive of a scrotal or testicular mass.
- In the evaluation of abnormal semen findings, testicular biopsy should be reserved for patients with normal results of hormonal studies and azoospermia to evaluate for obstruction or congenital absence of the vas and possible surgical repair or for possible use of in vitro fertilization with intracytoplasmic sperm injection.

Diagnosis and Treatment

An overall summary of clinical and laboratory findings, potential diagnoses, and recommended evaluation or treatment strategies in adult male patients with hypogonadism is presented in the table below.

Table. Summary of Findings, Potential Diagnoses, and Recommended Strategies in Adult Male Patients with Hypogonadism*

Testicular size**	FSH	LH	Testosterone***	Semen analysis	Diagnosis	Eva Tr
Not palpable	increased	increased	decreased	Azoospermia	Anorchism	ex
Not palpable	increased	increased	N*** or decreased	Azoospermia	Bilateral cryptorchidism	ex
< 5 mL	decreased	decreased	decreased	Azoospermia, oligospermia	Kallmann's syndrome, hypogonadotropic hypogonadism	T for hC FSH sper
< 5 mL	increased	increased	N*** or decreased	Azoospermia	Klinefelter's syndrome, other hypergonadotropic syndromes	Ka cor v
8 to 15 mL	increased	N	N	Azoospermia, oligospermia	Germinal damage: toxins, idiopathic	Fe wi
10 to 20 mL	decreased	decreased	decreased	Oligospermia	Adult acquired hypogonadotropic hypogonadism	Pitu prol d

Testicular size**	FSH	LH	Testosterone***	Semen analysis	Diagnosis	Eva Tr
						oth as s
10 to 20 mL	N or increased (variable)	N or increased (variable)	N*** or decreased	Variable	Senescence	T if s wi
15 to 20 mL	N or increased	N	N	Oligospermia	Varicocele, drugs, idiopathic	vari if v Opti fact
Variable phenotype	increased (variable)	increased (variable)	increased	Variable	T receptor defects, Reifenstein's syndrome	(de m surg
20 to 30 mL	N	N	N	Azoospermia	Obstruction	Ferti repa

* FSH = follicle-stimulating hormone; GnRH = gonadotropin-releasing hormone; hCG = human chorionic gonadotropin; hMG = human menopausal gonadotropin; ICSI = intracytoplasmic sperm injection; IUI = intrauterine insemination; IVF = in vitro fertilization; LH = luteinizing hormone; MRI = magnetic resonance imaging; N = normal; SHBG = sex hormone-binding globulin; T = testosterone.
** Normal testicular size is 20 to 30 mL. Testicular size is used here as a clinical finding to help narrow the differential diagnosis. Some variation beyond the listed ranges may exist for a specific condition. Use of this variable is optional; the diagnosis should be based on the total clinical picture.
*** Because of changes in SHBG levels, total testosterone may be in the normal range in the setting of low testosterone production. Level of SHBG or free testosterone should be used in this setting to determine whether treatment options should be considered.

Testosterone Therapy in Adult Male Patients with Hypogonadism

Ideally, testosterone therapy should provide physiologic range serum testosterone levels (generally between 280 and 800 ng/dL) and physiologic range dihydrotestosterone and estradiol levels, which would allow optimal virilization and normal sexual function. Testosterone therapy can be used in the male patient with hypogonadism who is not interested in fertility or not able to achieve fertility.

In late teenage male patients with delayed puberty, testicular size should be monitored for evidence of onset of puberty. In this setting, short-term, low-dose testosterone therapy should be withdrawn to determine whether spontaneous puberty is occurring.

The following preparations of testosterone have been approved by the Food and Drug Administration (FDA) for clinical use (refer to the original guideline document for details on dosage, scheduling, and preparation):

- Long-acting intramuscular preparations
- Short-acting intramuscular preparations
- Scrotal patches
- Transdermal patches
- Transdermal gel
- Orally administered agents

Monitoring Issues and Side Effects of Testosterone Therapy

Periodic follow-up of patients receiving testosterone therapy is needed. During the first year of testosterone therapy, the clinical response and the side effects should be monitored at 3- to 4-month intervals.

For patients receiving testosterone injections, a serum testosterone level should be measured at the midpoint between injections to ensure that the value is near the middle of the normal range. Most patients using testosterone gel have constant blood levels of testosterone over 24 hours; thus, the time of measurement is usually not critical. The testosterone patch preparations usually yield peak serum testosterone values within 4 to 8 hours after application.

Examination of the prostate should be done routinely, along with a prostate-related symptom assessment every 6 to 12 months. Prostate-specific antigen (PSA) levels should be determined annually in older men receiving testosterone replacement therapy. High PSA levels should be further evaluated with a highly specific PSA assay, if available. If results are abnormal, a urologic consultation should be sought, and testosterone replacement therapy should be terminated. Men receiving testosterone replacement therapy and finasteride should be considered for further evaluation even with PSA values in the upper normal range. Testosterone treatment should not be administered to men with high PSA values or significantly increasing PSA levels. Testosterone, and especially dihydrotestosterone, stimulates growth of the prostate and seminal vesicles.

Gynecomastia may result from the aromatization of testosterone to estradiol and changes in SHBG levels. Surgical therapy may be considered for some patients. Men with a genetic susceptibility to alopecia may note worsening of this problem with testosterone therapy.

Testosterone stimulates the bone marrow production of erythrocytes. The result is an increased hematocrit in some men, with the possibility of hyperviscosity side effects. The hematocrit should be determined every 6 months for the first 18 months and then yearly thereafter if it is stable and normal. Testosterone therapy should be decreased or discontinued if the hematocrit increases to above 50%.

Lipid disturbances in testosterone-treated male patients are generally not a problem because of the aromatization of testosterone to estradiol. The ratio of high-density lipoprotein (HDL) to total cholesterol generally remains constant. Anabolic steroids, used in oral testosterone preparations, that are not aromatized increase low-density lipoprotein (LDL) and lower high-density lipoprotein levels and thus could increase cardiovascular risk. An initial lipid profile should be recorded, and a follow-up profile should be obtained after 6 to 12 months of therapy and then yearly thereafter.

Sleep apnea may also be a problem in some men, and testosterone therapy should be discontinued until the sleep apnea problem can be adequately addressed. The patient should be asked about fatigue during the day in addition to disordered sleep. A sleep study should be done if symptoms are present.

Because pharmacologic use of testosterone will suppress spermatogenesis, the use of testosterone preparations may substantially reduce fertility in otherwise normal men. This adverse effect is often an issue with the illicit use of testosterone.

Gonadal Stimulation in Hypogonadotropic Hypogonadism

Because gonadotropin or GnRH therapy is effective only in hypogonadotropic hypogonadism, this diagnosis must be firmly established before consideration of therapy. Although these agents may also be used to induce puberty in boys and to treat androgen deficiency in hypogonadotropic hypogonadism, the major use of these preparations is in the initiation and maintenance of spermatogenesis in hypogonadotropic men who desire fertility.

Gonadotropin Therapy in Androgen Deficiency

It is known that hCG binds to Leydig cell LH receptors and stimulates the production of testosterone. Peripubertal boys with hypogonadotropic hypogonadism and delayed puberty can be treated with hCG instead of testosterone to induce pubertal development. The initial regimen of hCG is usually 1,000 to 2,000 IU administered intramuscularly two to three times a week. The clinical response is monitored and testosterone levels are measured about every 2 to 3 months. Dosage adjustments of hCG may be needed to determine an optimal schedule. Increasing doses of hCG may reduce testicular stimulation by down-regulating the end-organ; thus, a more optimal result may occur with less frequent or reduced dosing. The half-life of hCG is long.

Gonadotropin Therapy for Induction of Spermatogenesis

Male patients with onset of hypogonadotropic hypogonadism before completion of pubertal development may have testes smaller than 5 mL. These patients usually require therapy with both hCG and human menopausal gonadotropin (or FSH) to induce spermatogenesis. Men with partial gonadotropin deficiency, or who have previously (peripubertally) been stimulated with hCG, may initiate and maintain production of sperm with HCG only. Men with postpubertal-acquired hypogonadotropic hypogonadism and who have previously had normal production of sperm can also generally initiate and maintain production of sperm with hCG therapy only. Fertility may be possible at sperm counts much lower than what

would otherwise be considered fertile. Counts of less than 1 million/mL may be associated with pregnancies under these circumstances. It is imperative that the female partner undergo assessment for optimal fertility before or concurrently with consideration of therapy in the man.

Therapy with hCG is generally begun at 1,000 to 2,000 IU intramuscularly two to three times a week, and testosterone levels should be monitored monthly to determine whether any therapeutic adjustments are needed to normalize the levels. It may take 2 to 3 months to achieve normal levels of testosterone. When normal levels of testosterone are produced, examinations should be conducted monthly to determine whether any testicular growth has occurred. Sperm counts should also be conducted monthly during a 1-year period. Because of the high cost of human menopausal gonadotropin (or FSH) preparations, hCG should be the initial therapy of choice for at least 6 to 12 months. Use of hCG, in the absence of exogenous FSH, can often complete spermatogenesis in men with partial gonadotropin deficiency. In general, the response to hCG can be predicted on the basis of the initial testicular volume—the greater the initial testicular volume, the greater the chance of responding to hCG only.

If spermatogenesis has not been initiated by the end of 6 to12 months of therapy with hCG or LH, administration of an FSH-containing preparation is initiated in a dosage of 75 IU intramuscularly three times a week along with the hCG injections. After 6 months, if sperm are not present or are present in very low numbers (<100,000 per mL), the human menopausal gonadotropin (or FSH) dosage can be increased to 150 IU intramuscularly three times a week for another 6 months. If pregnancy occurs, the patient´s regimen can be switched to only hCG to allow continued spermatogenesis for subsequent potential pregnancies. After delivery, if no further pregnancies are desired, the patient can be switched to testosterone therapy if desired, or long-term hCG therapy can be continued in conjunction with appropriate contraceptive measures, if needed. Rarely, antibodies against hCG may arise and prevent any response to therapy; in such a case, human LH may be effective. Recombinant LH has recently become available and may be of use in selected patients.

Gonadotropin-releasing Hormone (GnRH) Therapy

In patients with an otherwise intact pituitary gland and hypogonadotropic hypogonadism, synthetic gonadotropin-releasing hormone (GnRH) can be given in a pulsatile fashion subcutaneously through a pump every 2 hours. GnRH therapy is monitored by measuring LH, FSH, and testosterone levels every 2 weeks until levels are in the normal range, at which point monitoring can be adjusted to every 2 months. GnRH can be used to initiate pubertal development, maintain virilization and sexual function, and initiate and maintain spermatogenesis. In most patients, these effects may take from 3 to 15 months to achieve sperm production. As with gonadotropin therapy, fertility can be achieved with very low sperm counts—often in the range of 1 million/mL. GnRH may be more effective than gonadotropin stimulation in increasing testicular size and initiating spermatogenesis in many patients with hypogonadotropic hypogonadism.

Other Treatment Considerations

Antiestrogen Therapy in Oligospermia

Currently, the guideline authors do not recommend the general use of clomiphene citrate or tamoxifen for treatment of oligospermia in male patients.

Assisted Reproductive Technology

The ability to perform in vitro fertilization with intracytoplasmic sperm injection directly into the egg has revolutionized the approach to male subfertility. A single sperm or immature form retrieved from the testicle is sufficient to fertilize an egg and provide a reasonable chance at pregnancy. In vitro fertilization with intracytoplasmic sperm injection may be a viable option in many men with hypogonadism who cannot otherwise be induced to produce enough sperm to result in pregnancy as well as in the presence of a female factor that may further make pregnancy by the couple difficult or impossible. The procedure is expensive and seldom covered by health insurance; therefore, this technology will generally not replace conventional gonadal stimulation protocols. Intrauterine insemination may also be a low-cost option in suitable women when the man has mild to moderate oligospermia.

Pituitary Tumors

Patients with acquired hypogonadotropic hypogonadism may require assessment for a possible pituitary tumor with appropriate pituitary imaging studies, such as MRI, and determination of a prolactin level. Depending on the presence or absence of a tumor, other hormonal testing may be indicated, including thyroid and adrenal function tests. Further evaluation and treatment options would depend on what hormonal deficits are present, the size and site of the tumor, the operability of the tumor, and the patient´s preferences in specific circumstances.

If a prolactinoma is present, therapy would be directed toward correcting this problem before initiation of other therapy. Medical therapy with bromocriptine, pergolide, or cabergoline may effectively reduce prolactin levels sufficiently to allow gonadal function to resume or allow stimulation with gonadotropins. Even when prolactin levels cannot be normalized, hCG therapy alone or in conjunction with human menopausal gonadotropin (or FSH) therapy may stimulate spermatogenesis in treated prolactinomas and result in pregnancies.

Surgical therapy should especially be considered for significant pituitary tumors that are not prolactin-secreting microadenomas. Surgical treatment may also be an option in prolactin-secreting microadenomas if patients have severe side effects from medications or prefer this approach after being appropriately informed of the risks and benefits of medical versus surgical management.

Gynecomastia

Many men have psychological problems resulting from gynecomastia. This problem should be taken seriously and discussed with the patient. Use of aromatase inhibitors, such as testolactone, has been tried but has yielded limited benefit. A breast reduction surgical procedure is often required for psychological well-being.

Psychological Counseling

Men with hypogonadotropic disorders frequently have associated mood disturbances, including depression, aggression, poor self-esteem, and learning problems. In such cases, psychological counseling is often needed to allow proper identification and treatment of these problems. Counseling should also include significant others, if possible.

CLINICAL ALGORITHM(S)

None provided

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

Most of the content in the guideline is based on literature reviews. In areas of uncertainty, professional judgment was applied.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

These guidelines on the evaluation and treatment of hypogonadism in adult male patients represent not only a source of guidance for health-care professionals but also an appeal to clinicians to increase their awareness of the problem and to discuss these issues with their at-risk patients. With growing awareness and increased research efforts, both the duration and the quality of life for aging men will improve.

Treatment of hypogonadism may:

- Restore sexual function, libido, well-being, and behavior
- Produce and maintain virilization
- Optimize bone density and prevent osteoporosis
- Possibly normalize growth hormone levels in elderly men
- Possibly affect the risk of cardiovascular disease
- Restore fertility in cases of hypogonadotropic hypogonadism

POTENTIAL HARMS

Cardiovascular Risk

- Currently, whether testosterone replacement therapy in men with hypogonadism increases, decreases, or has a neutral effect on cardiovascular risk remains uncertain.
- A potential risk of testosterone therapy is the propensity of testosterone to increase platelet aggregation and thrombogenicity.

Side Effects of Testosterone Therapy

- Gynecomastia

- Increased hematocrit with the possibility of hyperviscosity side effects
- Sleep apnea
- Worsening alopecia in those with a genetic susceptibility
- Skin irritation with transdermal patch use
- Reduction of fertility in otherwise normal men

CONTRAINDICATIONS

CONTRAINDICATIONS

Contraindications to Testosterone, Gonadotropin-releasing Hormone (GnRH), and Gonadotropin Therapy

Testosterone replacement, pulsatile GnRH therapy and gonadotropin therapy are contraindicated in men with prostate cancer, male breast cancer, or untreated prolactinoma. Treatment with these medications can stimulate tumor growth in androgen-dependent neoplasms. Careful examination of the male breast and prostate is required initially and at follow-up visits. In addition to prostate examination, baseline and follow-up prostate-specific antigen (PSA) levels should be determined in older men at increased risk for prostate cancer. Men with symptomatic prostatism should undergo evaluation and treatment for this problem before testosterone replacement therapy is considered. Sleep apnea and polycythemia, which may cause hyperviscosity, are relative contraindications to the use of testosterone therapy. Testosterone treatment will tend to reduce sperm counts and testicular size and should not be used in men currently seeking fertility.

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

- These guidelines are a working document that reflects the state of the field at the time of publication. Because rapid changes in this area are expected, periodic revisions are inevitable. The guideline developers encourage medical professionals to use this information in conjunction with their best clinical judgment. The presented recommendations may not be appropriate in all situations. Any decision by practitioners to apply these guidelines must be made in light of local resources and individual patient circumstances.
- Of prime consideration is the paucity of long-term research studies of the identification of men at risk for complications related to a decreased testosterone level, optimal assessment of such patients, optimal treatment, and potential complications relating to long-term therapy.
- Currently, whether testosterone replacement therapy in men with hypogonadism increases, decreases, or has a neutral effect on cardiovascular risk remains uncertain. Long-term prospective research must be conducted to assess the role of endogenous testosterone and testosterone replacement therapy on cardiovascular risk in men. No specific recommendations on this issue are possible until further research clarifies the potential risks and benefits of therapy.

- No clear relationship has been established between testosterone replacement therapy and prostate cancer, although anecdotal reports have been published. Long-term studies are needed to clarify this issue.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Living with Illness

IOM DOMAIN

Effectiveness
Patient-centeredness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

AACE Hypogonadism Task Force. American Association of Clinical Endocrinologists medical guidelines for clinical practice for the evaluation and treatment of hypogonadism in adult male patients--2002 update. Endocr Pract 2002 Nov-Dec;8(6):439-56. [77 references]

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

1996 (revised 2002)

GUIDELINE DEVELOPER(S)

American Association of Clinical Endocrinologists - Medical Specialty Society
American College of Endocrinology - Medical Specialty Society

SOURCE(S) OF FUNDING

American Association of Clinical Endocrinologists (AACE)

GUIDELINE COMMITTEE

American Association of Clinical Endocrinologists (AACE) Hypogonadism Task Force

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

Task Force Members: Steven M. Petak, MD, JD, FACE, Chairman; Howard R. Nankin, MD, FACE; Richard Spark, MD, FACE; Ronald S. Swerdloff, MD; Luis J. Rodriguez-Rigau, MD, FACE

FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Not stated

GUIDELINE STATUS

This is the current release of this guideline.

It updates a previous version: American Association of Clinical Endocrinologists (AACE), American College of Endocrinology (ACE). AACE clinical practice guidelines for the evaluation and treatment of hypogonadism in adult male patients. Jacksonville (FL): AACE; 1996. 28 p. (AACE clinical guidelines; no. 1996).

GUIDELINE AVAILABILITY

Electronic copies: Available in Portable Document Format (PDF) from the American Association of Clinical Endocrinologists (AACE) Web site.

Print copies: Available from the American Association of Clinical Endocrinologists, 1000 Riverside Ave., Suite 205, Jacksonville, FL 32204.

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

This summary was completed by ECRI on October 1, 1998. The information was verified by the guideline developer on December 1, 1998. This summary was updated by ECRI on February 27, 2003. The information was verified by the guideline developer on March 24, 2003.

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